Editorials

Power on the Demand Side of Health Care

SINCE WORLD WAR II this nation has made health and health care top priorities. Experience during the war made it evident that medicine and medical care could really make a difference and after the war medical research was wisely and generously supported by our government. Medical progress was substantial and well publicized. Soon it became a national objective, not only to continue this enormously productive research, but to make its results more readily available for the benefit of all the people. Since the mid-1960s there have been massive programs to make health care available to all by developing and modernizing health care institutions, increasing the numbers of health professionals and providing third party coverage of the costs through health insurance in the private sector and government programs to assure care for needy children, the elderly and the poor. And, most recently, health and fitness has turned out to be something of a national obsession, and a booming business for those who make and sell the supplies and know-how for it. The supply side of health and health care has clearly made great strides, and the rising costs of all this are familiar enough.

While the emphasis has so far been mostly on the supply side, there has also been a growing awareness among the public of the value of health and health care, and their importance to well-being, quality of life and, yes, to prolongation of life. This interest is giving rise to a new demand side for health care whose power to affect what is done about costs has hardly begun to be felt. So far the public does not seem to be as concerned about health care costs as are those on the supply side in both the private and public domain, who are promoting an atmosphere of crisis in health care costs. The public has not yet really spoken on health care costs. However, there is no doubt that the public is impressed with the evidence that medical control of hypertension can reduce strokes, that many forms of cancer (to which they may be susceptible) can now be effectively treated, that such things as coronary bypass operations and hip replacements can enormously improve quality of life, and perhaps even its length. The possibilities of readily available transplants or prosthetic replacements for worn or defective parts excite the imagination to say the least. The public perception is clearly that modern medical care is something worth having. This will surely lead to a greater exercise of power on the demand side of health care, and this will serve to counterbalance the present exercise of power on the supply side, although to an extent that is now difficult to predict.

And where is this power on the demand side to be found? For one thing, as costs are cut on the supply side to the detriment of patient care, these issues are being taken to court, and the courts tend to side with a sick or injured person who has been deprived. It is of note that the American Medical Association has recently decided to challenge, through legal action, the constitutionality of provisions of the Medicare amendments passed with the Budget Deficit Reduction Act of 1984 because they allegedly limit access of patients to care. Special interest groups, such as those who support the right-to-life movement, have already made their power felt on the demand side of health care. And at the other end of the life spectrum, the power of the American Association of Retired Persons (AARP) is beginning to be felt in the political arena of health care. And in the not too distant future, the number of veterans seeking health care is expected to rise sharply—and the power of the veterans' lobby to support its own system of health care is well known. In short, there is real power on the demand side of health care.

One senses that the battle between the supply side and the demand side has yet to be fully joined. Power on each side seems to be of a somewhat similar order of magnitude. How the problem of escalating costs is to be solved in this predictable struggle remains to be seen. It is clear that the opportunities for health and health care and the problems of costs will not go away. One cannot help wondering if cooperation among all those concerned may not be a better answer than the competition and confrontation that now seem to be the order of the day. But in any case, the medical profession will always be found in the patient's corner and on the side of better health and better health care for all our citizens. We will have it no other way.

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Sudden Cardiac Death—A Perspective

DESPITE A progressive decline in the death rate from cardiovascular disease over the past decade, sudden (arrhythmic) cardiac death remains an imposing challenge, accounting for at least 50% of all cardiac deaths.[1] Recent investigations advancing our understanding of the epidemiology and mechanisms of this problem have been paralleled by improvements and new developments in multiple therapeutic methods, as outlined in the transcript of the UCLA conference elsewhere in this issue.

The title of this conference, "Recent Trends in the Management of Life-Threatening Ventricular Arrhythmias," is broad and merits comment. We now recognize many categories of ventricular arrhythmias. While ventricular fibrillation and sustained ventricular tachycardia are obviously life-threatening arrhythmias, ventricular ectopy, whether complex or simple, is not a life-threatening arrhythmia. Additionally, even most episodes of nonsustained (3 beats to 30 seconds) ventricular tachycardia are asymptomatic and not necessarily life-threatening. Although nonsustained arrhythmias and ventricular ectopy may in some patients be markers of risk for arrhythmic death, the context in which the arrhythmia occurs—that is, the presence, type and severity of underlying heart disease—significantly influences this risk. The most common anatomic substrate associated with sudden death is obstructive coronary artery disease resulting in a previous myocardial infarction, and the vast majority of clin-

This study was supported in part by grants from the American Heart Association, Southeastern Pennsylvania Chapter, Philadelphia, and National Heart, Lung and Blood Institute, Bethesda, Md (HL28093, HL00361, HL24278).

ical and experimental studies examining risk factors, mechanisms and treatment of sudden death have been done in patients with atherosclerotic coronary artery disease. It is premature to assume that similar mechanisms are responsible for sudden death in noncoronary disease. Therefore, in other diseases associated with significant risk for sudden death such as cardiomyopathy, other risk factors may be more appropriate to guide therapy,[2] and different pharmacologic agents may be necessary to prevent this event.

Central to the current approach to recognition, prevention and treatment of sudden cardiac arrest has been the realization that two major perturbations in the course of chronic coronary artery disease may initiate this event: acute, severe ischemia resulting in ventricular fibrillation, and ventricular tachycardia occurring without any apparent provocative factor. The latter appears to be more important statistically, as a few patients dying suddenly show pathologic evidence of acute myocardial necrosis, and most survivors of cardiac arrest do not evolve electrocardiographic or enzymatic evidence of myocardial infarction, nor do they give a history of ischemic symptoms immediately preceding the acute event.

A large body of evidence supports reentry as the most common mechanism underlying ventricular tachycardia in the course of chronic coronary artery disease. For reentry to occur there must be a myocardial substrate with appropriate anatomic and physiologic characteristics, which is activated by appropriate triggers. In the case of ventricular tachycardia, the triggers are usually spontaneous ventricular premature depolarizations and the substrate is an area(s) of abnormal tissue on the border of an old myocardial infarction. If this schema is correct, patients at risk of sustained arrhythmias should be identifiable by the presence of appropriate triggers (using Holter monitoring) or by the presence of a substrate. Identification of a latent substrate has been more difficult, but newer techniques such as programmed stimulation and the signal averaged electrocardiogram[3] may permit this. Similarly, therapy for ventricular tachyarrhythmias could be approached by either eradicating the triggers pharmacologically (the efficacy of which is judged by suppression of spontaneous ectopy on Holter monitors) or disabling the substrate pharmacologically or surgically. The use of programmed stimulation to evaluate antiarrhythmic therapy is based on the ability of conventional antiarrhythmic agents and map-guided surgical treatment to eradicate inducible arrhythmias. Implicit in this model of reentrant arrhythmias is the hypothesis that a therapeutic modality must be effective against only the trigger or the substrate to prevent spontaneous arrhythmias. A corollary of this is that a pharmacologic antiarrhythmic agent that disables the reentrant circuit need not be similarly efficacious in eradicating spontaneous ectopy, and vice versa.[4] A major problem in guiding therapy through suppression of spontaneous ectopy is the remarkable degree of random variation and spontaneous ectopy that many patients show, making interpretation of true therapeutic effects very difficult. In addition, 20% to 25% of patients with documented sustained tachyarrhythmias have only rare and noncomplex spontaneous ectopy. Finally, as only one properly timed ectopic beat is often needed to initiate a tachycardia, it becomes very difficult to completely suppress ectopy without producing intolerable drug side effects. Thus, we have placed greater reliance recently on eradicating the reentrant substrate as the appropriate endpoint for pharmacologic and surgical therapy. Electrophysiologic studies may be helpful not only in identifying drugs capable of preventing inducible arrhythmias, but are equally useful in predicting ineffective drug regimens. By identifying ineffective drugs under safely controlled catheterization laboratory conditions, the risks of empiric trials whose endpoint for failure may be out-of-hospital cardiac arrest are minimized.

Much attention is given in the accompanying symposium to the investigational antiarrhythmic agent, amiodarone. The rosy picture painted of the use of this agent must be tempered by the realization that there are many problems associated with its use. Several large series that have examined the efficacy of this agent in patients having cardiac arrest or sustained ventricular tachycardia have shown that 70% to 80% of patients are free from recurrent arrests or sustained arrhythmias.[5-10] However, in each series side effects (some potentially fatal) necessitating discontinuation of the drug regimen occurred in almost one out of every five patients.[5-10] Thus, only 50% to 60% of patients continue taking the drug and are free of arrhythmias and side effects. Although the drug has no direct negative inotropic effects, it is a noncompetitive adrenergic blocker and has been associated with worsening of congestive heart failure.[6,8,9]

Amiodarone appears to be unique not only in its high degree of efficacy in patients whose arrhythmias have been resistant to conventional agents, but it also appears to work by a different, but as yet unclear, mechanism. In contrast to results with type I antiarrhythmic agents, the ability of amiodarone to suppress ventricular tachycardia induced by programmed stimulation does not appear to correlate with its effects on spontaneous recurrences. The reason for this is not clear at present. Although this drug's effects on intracellular electrophysiologic variables (the primary lengthening of refractoriness) differ from those of type I antiarrhythmic agents, it is not at all clear that this explains its efficacy. In fact, amiodarone does affect the sodium channel and therefore under certain conditions may greatly alter the upstroke of the cardiac action potential, an effect which itself may explain alterations in the tachycardia rate.[11] It is tempting to speculate that the potent suppression of spontaneous ectopy shown by this drug plays a role by interfering with the trigger mechanism for reentrant tachycardias. However, in contrast to the data accumulated by the group at UCLA, not all investigators have shown that suppression of spontaneous ectopy by amiodarone predicts clinical efficacy.[9] Furthermore, the crude clinical measurements available to us at present do not permit us to assume that alterations in arrhythmias such as prolongation of cycle length are necessarily due to specific intracellular effects of drugs such as lengthening of refractoriness. The long duration of action of amiodarone, while beneficial in permitting once a day dosing, has associated problems: the onset of action is delayed and its effects may be prolonged for weeks after the drug is discontinued—that is, when serious side effects develop.

Thus, although we have seen numerous advances in our understanding of "life-threatening arrhythmias," we have far to go. Our understanding of the mechanisms underlying these arrhythmias is incomplete. Although we know slow conduction is a requirement for reentry, a number of conditions may contribute to slowed conduction. For example, while slow

response cells may predispose to slow conduction and these have been found in areas of previous myocardial infarction, most experimental and clinical evidence suggests that cell-to-cell uncoupling rather than intracellular abnormalities are responsible for the slow conduction predisposing to reentry in patients with myocardial infarction.[12,13] Our understanding of the mechanisms by which antiarrhythmic drugs work is incomplete. Studies of the effects of drugs on cellular action potentials alone are inadequate to explain in vivo drug actions in nonhomogeneous infarcted myocardium in which complex reentrant circuits may involve cells with varying degrees of abnormality. Additionally, the conventional classification of antiarrhythmic drugs, as described in this symposium, fails to take into account use-dependent properties of these agents.[14] We are still in need of specific and sensitive noninvasive predictors of patients at risk for cardiac arrest. We have yet to identify the ideal broad-spectrum antiarrhythmic agent that is inexpensive, requires infrequent dosing and is highly efficacious and free of side effects. Electrical therapies for ventricular arrhythmias such as implanted cardioverter-defibrillators require many technologic improvements. Furthermore, these devices are less than optimal in that they do not prevent the occurrence of arrhythmias. Surgical therapy is still evolving, and on the horizon are potentially less invasive therapies such as catheter ablation of arrhythmogenic foci. However, all of these efforts may fail to achieve our most desirable endpoint—improvement in duration and quality of life—if ways are not found to salvage damaged myocardium and improve ventricular function.

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Physicians' Incomes

ACCORDING TO THE AMA Center for Health Policy Research, physicians' average net incomes reached $106,300 last year. The $100,000 figure has been bruited about for some time in the press and elsewhere. And somehow there is an implication that physicians should feel guilty, as though individually or collectively they had done something wrong. While there is no doubt that many physicians have incomes substantially less than this, others—rightfully or wrongly depending on one's point of view—have incomes that are substantially larger. But perhaps the question is, how do physicians' salaries compare with training and responsibilities?

In spite of all the talk—and all the laws—about informed consent and patient responsibility, a patient's life or quality of life is often placed by the patient in the hands of a physician. This is obvious when a patient has given informed consent and is unconscious and being operated on by a surgeon, but it is also often true when a diagnosis is made, or a risky treatment is undertaken, or sometimes even when an x-ray film or a laboratory test is interpreted by a physician specialist in an emergent situation. Where else in our society is there anything like a comparable situation?

Airplane pilots who operate large commercial jet aircraft come to mind. Much as they do with their physicians or surgeons, people place their lives and future well-being in the hands of commercial airplane pilots, and expect them to have the competence and training to do the job, and to exercise expert judgment in the event that something should go wrong.

It is rumored that the salaries of commercial airplane pilots are now approaching if not surpassing $100,000 a year. Is this too in some way wrong? Is it too much? Or are we in fact uncovering something like a going rate of pay for those professionals who have the skill, training, experience and stamina to assume this kind of direct personal responsibility for the lives and future well-being of other people in what can sometimes turn out to be very complex and critical situations? The compensation of physicians, in terms of skill, training, experience and stamina for direct personal responsibility for the lives and well-being of others who are under their care, could become an issue of increasing concern as we find ourselves propelled faster and further into what Eli Ginzberg has called the monetarization of patient care.[1]

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